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Published in:
American Naturalist

DOI:
10.1086/674008

The Evolution of Age-Dependent Plasticity

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Submitted October 19, 2012; Accepted July 12, 2013; Electronically published November 12, 2013

Online enhancement: appendix. *Dryad data:* http://dx.doi.org/10.5061/dryad.00nd1.

ABSTRACT: When organisms encounter environments that are heterogeneous in time, phenotypic plasticity is often favored by selection. The degree of such plasticity can vary during an organism's lifetime, but the factors promoting differential plastic responses at different ages or life stages remain poorly understood. Here we develop and analyze an evolutionary model to investigate how environmental information is optimally collected and translated into phenotypic adjustments at different ages. We demonstrate that plasticity must often be expected to vary with age in a nonmonotonic fashion. Early in life, it is generally optimal to delay phenotypic adjustments until sufficient information has been collected about the state of the environment to warrant a costly phenotypic adjustment. Toward the end of life, phenotypic adjustments are disfavored as well because their beneficial effects can no longer be fully reaped before death. Our analysis clarifies how patterns of age-dependent plasticity are shaped by the interplay of environmental uncertainty, the accuracy of perceived information, and the costs of phenotypic adjustments with life-history determinants such as the relative strengths of fecundity and viability selection experienced by the organism over its lifetime. We conclude by comparing our results with expectations for alternative mechanisms, including developmental constraints, that promote age-dependent plasticity.

Keywords: developmental plasticity, plasticity windows, reaction norms, eco-evo-devo, information sampling, dynamic optimization.

Introduction

Phenotypic plasticity is a universal property of living organisms (Tollrian and Harvell 1999; West-Eberhard 2003). Plasticity reveals itself as the capacity of a single genotype to produce different phenotypes in response to environmental influences during development. The adaptive use of information about environmental conditions distinguishes phenotypic plasticity from stochastic switching or bet hedging (Slatkin 1974), which is a risk-spreading strategy that is frequently employed by microbes (Veening et al. 2008) to help ensure long-term survival in an unpredictably varying environment.

A plastic genotype has a selective advantage over a nonplastic one, if the former has a higher net fitness than the latter averaged over the environments that the organism can encounter (Bradshaw 1965; Levins 1968). Theoretical studies suggest that plastic genotypes are superior in variable environments when sufficiently reliable environmental cues are available and costs of plasticity are low (Via and Lande 1985; Van Tienderen 1991; Gomulkiewicz and Kirkpatrick 1992; Houston and McNamara 1992; Schlichting and Pigliucci 1995; Ernande and Dieckmann 2004).

The plastic adjustment of phenotypes can involve morphological modifications, adaptations of physiological and neural regulation, or behavioral changes. A well-known example of morphological reconstruction is found in *Daphnia* sp., with individuals adapting to environmental conditions by growing a protective helmetlike structure in response to the presence of predators (Tollrian 1990). Physiological plasticity is observed, for instance, in several closely related species of larks (family *Alaudidae*), which can adjust their basal metabolic rate to the ambient temperature (Tieleman et al. 2003). In the rat (and several other mammals), the level of maternal care (pup licking/grooming) received early in life has long-lasting effects on the responsiveness to stress, mediated by brain-specific DNA methylation and differential expression of stress hormone receptors in the central nervous system (Szyf et al. 2007). Finally, an example of behavioral plasticity is found in the spider *Parawixia bistriata*, which adjusts the size and structure of its web to the type of prey it expects to catch (Sandoval 1994).

If organisms were able to acquire full information about which phenotype is optimal in a given situation, and if adjustments would be cost free and could be realized without time lags, then we would expect to see organisms with unlimited plasticity. Such hypothetical organisms (sometimes called "Darwinian demons" after Law 1979) would

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Am. Nat. 2014. Vol. 183, pp. 108–125.
DOI: 10.1086/674008

express highly specialized phenotypes and constantly switch between them as their environments change in order to express optimal trait values for every possible environmental situation. This clearly is not what we see in nature. One reason for this is that plasticity generally comes at a cost. Morphological adjustments are likely to be associated with high construction costs and may be difficult to reverse (Brönmark and Miner 1992; Van Buskirk 2000; Callahan et al. 2008), whereas physiological and behavioral plasticity is usually mediated by a redirection of neuroendocrine and hormonal regulatory pathways. The latter is often considered to be less costly than morphological reconstruction but can be associated nevertheless with a number of costly (e.g., mobilization of energy and tissue nitrogen) and potentially risky (e.g., downregulation of the immune system) physiological processes (reviewed in Sapolsky et al. 2000; Sapolsky 2002; Badre and Wagner 2006).

Limits to plasticity are also illustrated by the observation that many organisms are more responsive to environmental perturbations during some ages or life stages than during others (e.g., Dufty et al. 2002; Hoverman and Relyea 2007). These patterns are observed to vary across species (Hoverman and Relyea 2007) and traits (e.g., Taborsky 2006; Arnold and Taborsky 2010; Kotrschal and Taborksy 2010; Segers and Taborsky 2012). For instance, bryozoans can grow defensive structures in response to chemical predator cues only early in their life (Harvell 1991), and in rats, persistent stress resistance can be induced by maternal care only if experienced in the first week after birth (Szyf et al. 2007). In freshwater snails (*Helisoma trivolvis*), the ability to build defensive structures against predatory water bugs extends well beyond sexual maturity, whereas a reversal of this trait is only possible during early ontogeny (Hoverman and Relyea 2007). Finally, as an example of a species exhibiting a prolonged high degree of plasticity in a morphological trait, we mention barnacles (*Balanus glandula*), which maintain a lifelong ability to grow and shrink legs used for suspension feeding in response to flow conditions (Marchinko 2003).

It is not yet understood which factors determine the diverse patterns of age-dependent plasticity across species and traits that are observed in nature. In general, changes in plasticity with age are expected if an organism does not have perfect information at birth but can improve its estimate of the environmental state by integrating information accumulated over a longer period of time (Dufty et al. 2002). Some theoretical work exists on the evolution of reversible plastic responses (Gabriel et al. 1999, 2005), but to our knowledge, the evolution of age-dependent phenotypic plasticity has not been systematically explored.

Here we study how plasticity is expected to change with age in an environment that varies stochastically over time. To this end, we calculate optimal patterns of age-dependent plasticity and examine how these depend on the rates of environmental fluctuations, the organism's life history, and the relative strengths of selection on different components of fitness. We model the process of information acquisition, which is crucial for decision making in uncertain environments (Real 1992; Dall et al. 2005), and consider different degrees of perception accuracy and plasticity costs.

Model

The definition of our model will be structured as follows: first, we focus on the environment, which we assume to be both stochastically fluctuating and partially predictable. We then describe how organisms can predict future conditions based on current and past observations of the state of the environment. Next, we explain how organisms adjust their phenotypes depending on the gathered information, given a reaction norm for age-dependent plasticity. As a final step, we specify how the fitness of a reaction norm is calculated and outline the optimization procedure for finding a reaction norm that maximizes fitness. Figure 1 provides a preview of how these steps coincide with life-cycle events in our model and serves as a reference for some of the notation that will be developed.

Fluctuations in the State of the Environment (Fig. 1; Step 1)

We consider a population of organisms living in a variable environment that changes stochastically from one reproductive season to the next. At each of these time steps, the environment can be in one of two discrete states, denoted A and B, representing different ecological conditions, such as high-flow and low-flow conditions in an aquatic environment. It should be understood that these two conditions in general do not need to represent a "good" and a "poor" environmental state, even though this latter distinction is common and important. In fact, we are primarily interested in situations in which the two different ecological conditions call for different phenotypic specializations, such that the fitness rank of phenotypes may change when the environment switches from one state to the other.

The lifetime reproductive success of an individual depends on the sequence of environmental states it experiences during its life. We denote this sequence by $\boldsymbol{E} = (E_1, E_2, \ldots, E_T)$, where $E_t =$ A or B represents the state of the environment at time t, and T is the maximum lifetime of individuals. For each individual, time is measured relative to the moment of its birth and expressed in discrete time units corresponding to one reproductive sea-

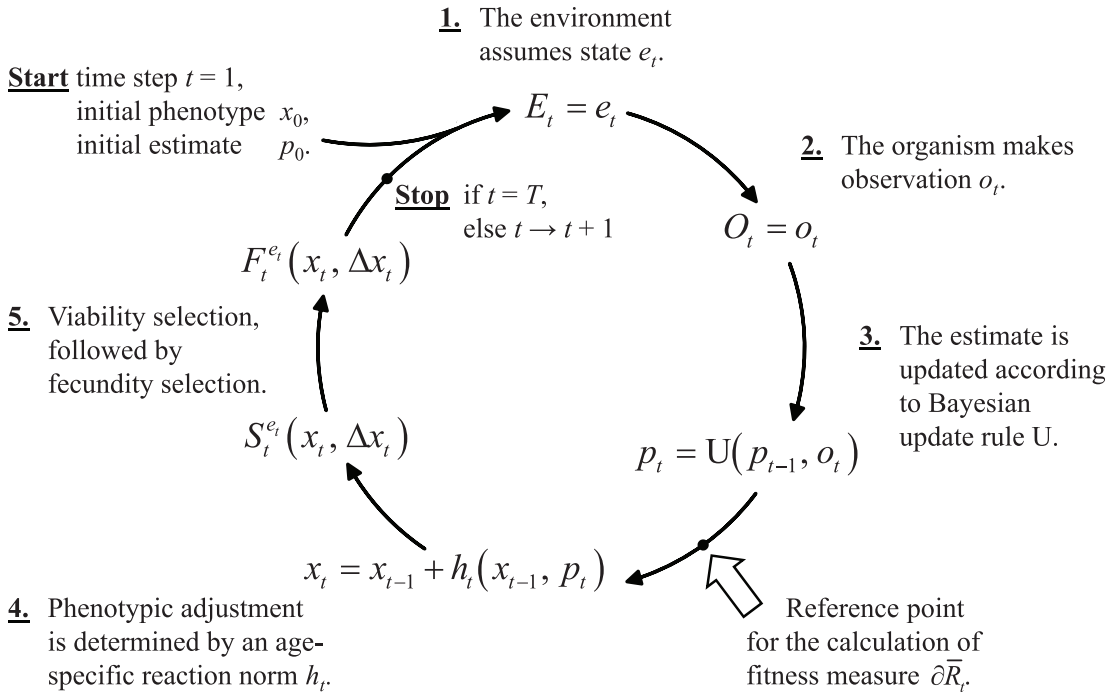

Figure 1: Order of events during a single time step. The sequence of events that occurs during a single time step starts with the determination of the environmental state (step 1). Each individual then samples the state of the environment (step 2) and uses the resulting observation to update its personal estimate p_t (step 3). Based on its new state, each individual then decides if and by how much it will adjust its phenotype (step 4). Viability selection acts at the end of each time step, potentially followed by the production of offspring. Both survival and fecundity are allowed to depend on the match between the current phenotype and the environment. In addition, in order to incorporate costs of plasticity, both fitness components may decrease as a function of the absolute size $\Delta x_t = |x_t - x_{t-1}|$ of the latest phenotypic adjustment step.

son. We assume that the state of the environment at time t is dependent on the environment's state at time $t - 1$, such that the E_t are correlated random variables. Accordingly, we model the environmental fluctuations as a first-order autoregressive stochastic process with two parameters, α and β, that define the rates of switching between environmental states. Specifically, α is the probability at each time step that the environment switches from state B to state A, which can be expressed as the conditional probability $\mathrm{P}[E_t = \mathrm{A}|E_{t-1} = \mathrm{B}]$. Likewise, β is the reverse transition probability, that is, $\beta = \mathrm{P}[E_t = \mathrm{B}|E_{t-1} = \mathrm{A}]$. Throughout, we focus on environments for which $0 < \alpha + \beta < 1$. Under this condition, E_t and E_{t+1} are positively correlated, such that given knowledge of the current state of the environment (E_t), the organism can predict the future state E_{t+1} and adjust its phenotype accordingly. The accuracy of this prediction is inherently limited, however, by the fact that E_t and E_{t+1} cannot be perfectly correlated in a changing (i.e., $\alpha + \beta > 0$) environment.

Environmental Sampling and the Integration of Information (Fig. 1; Steps 2, 3)

A second factor limiting an organism's ability to predict future conditions is that the state of the environment may not be directly observable, forcing individuals to infer information from a finite sample of imperfect cues. In our model, therefore, we introduce the random variable O_t (O_t = A or B) to represent the observation of the state of the environment made by an individual at time t. The observed and actual environmental states may be strongly or weakly correlated to each other, depending on the reliability of the information that is accessible to the organism. Specifically, we assume that irrespective of the state of the environment, observations are correct with probability a, such that $\mathrm{P}[O_t = o_t|E_t = e_t]$ is equal to either a or $1 - a$, depending on whether the current state of the environment is perceived correctly ($o_t = e_t$) or not ($o_t \neq e_t$). Here and henceforth, o_t (o_t = A or B) is used to denote the actual observation at time t made by a par-

ticular individual under consideration (i.e., o_t is a realization of O_t). A similar consistent use of upper- and lowercase symbols distinguishes between the state of the environment as a random variable (E_t) and its realization (e_t; see also notational conventions in the appendix; appendix available online). Throughout, we will refer to parameter a as the sampling accuracy.

Even though a single observation has limited accuracy, older individuals who have repeatedly sampled the environment may still be able to estimate the state of the environment reliably by integrating information over the sequence of observations $\boldsymbol{o}_t = (o_1, o_2, \ldots, o_t)$ they have made up to their present age. However, earlier observations are inherently less informative than more recent ones because the environment may have changed in the time since an observation was made. As a result, the organism needs to find a balance between rapidly discounting previous information so as to minimize the risk of making decisions based on out-of-date observations (adaptive forgetting; Kraemer and Golding 1997) and integrating over a large number of observations so as to avoid being misled by observation errors. An optimal solution for this problem is to use Bayesian updating after each observation in order to estimate how likely it is that the environment is currently in one state or the other.

Let us therefore assume that the organism is capable of keeping track of a state variable p_t that reflects its best possible estimate for the current state of the environment given the limited information to which it has access. As this information is fully contained in the sequence of observations, we define an individual's estimate p_t as a likelihood

$$p_t = \mathrm{P}[E_t = \mathrm{A} | O_t = o_t \cap O_{t-1} = o_{t-1} \cap \ldots \cap O_1 = o_1] \quad (1)$$

that is conditioned on the complete history of observations made by the individual up to its present age.

In the appendix, we derive how each individual can calculate its estimate p_t based on its prior knowledge of the state of the environment (represented by the previous estimate p_{t-1}) and its current observation o_t. This dependence can be expressed in the form of a Bayesian update rule U, which maps the previous estimate p_{t-1} to a new, updated estimate p_t after making observation $O_t = o_t$:

$$p_t = \mathrm{U}(p_{t-1}, o_t) = \begin{cases} \dfrac{a[(1-\beta)p_{t-1} + \alpha(1-p_{t-1})]}{a + (1-2a)[\beta p_{t-1} + (1-\alpha)(1-p_{t-1})]} & \text{if } o_t = \mathrm{A}, \\ 1 - \dfrac{a[\beta p_{t-1} + (1-\alpha)(1-p_{t-1})]}{a + (1-2a)[(1-\beta)p_{t-1} + \alpha(1-p_{t-1})]} & \text{if } o_t = \mathrm{B}. \end{cases} \quad (2)$$

The derivation of this result, which follows from Bayes's theorem and the laws of probability for conditionally independent events (appendix), rests on the assumption that the environmental switching rates and the sampling accuracy are "known" in the sense that the considered species has previously adapted to the considered fluctuating environment. As an implication, p_0, the initial estimate of a naive individual who has not yet made any observations, is taken to be equal to the long-term average frequency of environmental state A, $\mathrm{P}[E_t = \mathrm{A}] = \alpha/(\alpha + \beta)$.

Equation (2) conforms to the biological intuition in two ways. First, it confirms that prior information is less valuable in a more variable and less predictable environment. Specifically, in the absence of environmental autocorrelation ($\alpha = 1 - \beta$), knowledge of the previous state of the environment becomes useless for predicting the current state. Therefore, the right-hand side of equation (2) becomes independent of p_{t-1} if $\alpha = 1 - \beta$. Second, it indicates that the value of current information decreases with the frequency of observation errors in an individual's assessment of the environmental state. In the event that observations are as likely to be correct as not ($a = 1/2$), the right-hand side of equation (2) becomes independent of o_t. In that case, no information can be accumulated, and p_t remains at $p_0 = \mathrm{P}[E_t = \mathrm{A}] = \alpha/(\alpha + \beta)$.

In the typical situation considered in our analysis, the result of the Bayesian update rule depends on both the current, potentially erroneous observation and the information collected earlier. As an example of such a case, consider an organism in a fluctuating environment with $\alpha = 0.15$ and $\beta = 0.1$. With these switching rates, the long-term average frequency of environmental state A is $\alpha/(\alpha + \beta) = 0.6$, such that a naive organism does best by starting with an initial estimate $p_0 = 0.6$. Suppose that at age 1 the organism observes that the environment is in state B. On the basis of equation (2), it will then decrease its estimate p_1 to a value less than p_0 but larger than zero, because generally the organism cannot be certain that the environment truly is in state B based on this single observation. For instance, if the sampling accuracy is $a = 0.7$, we find $p_1 = 0.39$ (after observing B in this particular environment). Subsequent observations of environmental state B at ages 2 and 3 would further increase the organism's confidence that the environment is in state B (application of the Bayesian update rule gives $p_2 = 0.25$ and $p_3 = 0.18$). However, if the organism observes environmental state A at ages 4, 5, and 6, then the estimates go up again (in this case, eq. [2] gives $p_4 = 0.48$, $p_5 = 0.71$, and $p_6 = 0.83$).

The range of values that the estimate p_t can take is constrained by the inequalities $\alpha(1-a)/(2a-1) < p_t < 1 - \beta(1-a)/(2a-1)$ (this lower and upper bound is found by solving $p_t = \mathrm{U}(p_t, \mathrm{B})$ and $p_t = \mathrm{U}(p_t, \mathrm{A})$ for small α and β). Certainty about the state of the environment is

therefore inherently limited by both the environmental switching rates and the accuracy of individual observations. As a result, there is also a limit to an organism's knowledge gain through sampling.

Development of the Phenotype (Fig. 1; Step 4)

After the individual has sampled the environment and has integrated the newly obtained information with previous observations, it may adjust its phenotype. We allow the level of adjustment to depend on the organism's state, which encompasses its age, its phenotype at the previous time step, and its estimate of the state of the environment. For simplicity, we take the phenotype to be a one-dimensional trait that can take any value between 0 and 1 and describe its development by a recursion

$$x_t = x_{t-1} + h_t(x_{t-1}, p_t). \tag{3}$$

Here x_t denotes the phenotype at age t, and h_t is the reaction norm that captures how the organism adjusts its phenotype depending on its state after sampling the current environment. As for the estimate p_0, we assume that the initial phenotype x_0 has been set by adaptive evolution. Our further analysis, therefore, treats x_0 as an evolutionary trait that is optimized together with the reaction norm.

Given an initial phenotype x_0 and a reaction norm h, the recurrence relationship (3) and update rule (2) allow us to calculate an individual's developmental trajectory $x_0 \to x_1 \to \ldots \to x_T$ from the sequence of observations that the individual makes throughout its life (fig. 1). In the next section, we explain how the developmental trajectory determines an individual's lifetime reproductive success. As a final step, we outline the procedure for maximizing the expectation of this fitness measure over environmental states in order to find the optimal reaction norm.

Fitness Consequences of Plasticity (Fig. 1, Step 5)

The fitness of a reaction norm h depends on its average performance across all possible realizations of the sequence of environmental states. Moreover, in any given environment, not all individuals will make the same sequence of observations, due to errors in the assessment of environmental cues. As these errors can induce a change in the phenotypic trajectory, they represent an additional source of variation for the fitness of the reaction norm. Accordingly, the fitness function W, which has to be maximized to identify the optimal reaction norm, is defined by a double average

$$W = \prod_{\boldsymbol{e}} \Big(\sum_{\boldsymbol{o}} \mathrm{P}[\boldsymbol{O} = \boldsymbol{o} | \boldsymbol{E} = \boldsymbol{e}] R_1(\boldsymbol{o}, \boldsymbol{e}) \Big)^{P[\boldsymbol{E}=\boldsymbol{e}]}. \tag{4}$$

Here $R_1(\boldsymbol{o}, \boldsymbol{e})$ denotes the lifetime reproductive success (from age 1 onward) of an individual with observation sequence $\boldsymbol{o} = (o_1, o_2, \ldots, o_T)$ in environment $\boldsymbol{e} = (e_1, e_2, \ldots, e_T)$. The summation averages individual lifetime reproductive success over the distribution of observation sequences in environment $\boldsymbol{e}$, yielding the population-average fitness of the reaction norm in that environment. The product averages the population's fitness over all possible realizations of the environment $\boldsymbol{e}$ using the standard geometric mean fitness criterion for evaluating the long-term evolutionary success of a strategy in a stochastic environment (Lewontin and Cohen 1969).

All that remains to complete the definition of the model is to specify a procedure for determining $R_1(\boldsymbol{o}, \boldsymbol{e})$. One straightforward method is to calculate the expected reproductive success of an individual at age t and onward from the recursion $R_t(\boldsymbol{o}, \boldsymbol{e}) = S_t(F_t + R_{t+1}(\boldsymbol{o}, \boldsymbol{e}))$. Here S_t denotes the survival probability of the individual at age t, and F_t denotes its fecundity at that age. Iterating the recursion backward in time from $t = T$ to $t = 1$ (with the terminal reward $R_{T+1}(\boldsymbol{o}, \boldsymbol{e})$ defined to be zero) gives an expression for the lifetime reproductive success $R_1(\boldsymbol{o}, \boldsymbol{e})$.

From here on, fecundity and survival probability will be written as functions $F_t^{e_t}(x_t, \Delta x_t)$ and $S_t^{e_t}(x_t, \Delta x_t)$, respectively, to emphasize that these fitness components depend on the current environment $e_t (e_t = \text{A or B})$, the current phenotype x_t, and the phenotypic adjustment $\Delta x_t = |x_t - x_{t-1}|$ made by the individual at age t. The dependence on e_t and x_t is critical for modeling the benefits of plasticity (i.e., expressing a phenotype that matches with the environment), while the dependence on Δx_t is included to capture potential costs associated with the process of phenotypic adjustment. Our analysis excludes cases where an organism's current phenotype determines survival or fecundity later in life, as, for example, when the organism stores energy reserves for later use in reproduction. These more complex scenarios can be analyzed by introducing additional state variables, which we chose to avoid here.

Linearization of the Fitness Function and Evolutionary Optimization of the Reaction Norm

For any given fecundity and survival function, equation (4) can be maximized using evolutionary optimization methods (e.g., individual-based simulation). However, this approach provides limited biological insight. Therefore, we make a number of simplifying assumptions that enable us to obtain approximate expressions for the fitness function that clarify how the cost and benefit of plasticity interact with the life history of the organism. Here we only give a brief outline of this derivation; technical details are provided in the appendix. The main simplification is that we assume selection to be weak. This allows us to ignore, up

to first approximation, interaction effects between components of selection that are associated with different environmental states or act on different life-history stages. In addition, we take the costs of phenotypic adjustment to be independent of the state of the environment and first assume that $F_t^{e_t}$ and $S_t^{e_t}$ are linear in their arguments x_t and Δx_t before generalizing our results to arbitrary nonlinear functions in the appendix (see also fig. A1; figs. A1, A2 available online).

The first step in simplifying the fitness function is to consider an individual with a fixed phenotype $x_t = z$ and to use the average life history of this individual as a benchmark against which all fitness effects of plasticity are measured. If selection is weak, all fitness deviations from the reference life history are small, which implies that the environmental fluctuations have modest effects on survival and fecundity. With this in mind, we introduce two sets of (small) selection coefficients. First, the coefficients $f_t^{e_t} = (\partial/\partial x)F_t^{e_t}(x, 0)/\overline{F}_t$ and $s_t^{e_t} = (\partial/\partial x)S_t^{e_t}(x, 0)/\overline{S}_t$ quantify the relative difference in, respectively, fecundity and survival between two individuals whose phenotypes differ by one phenotypic unit. Positive values of these coefficients indicate that selection favors higher values of x_t in environment e_t. Second, the coefficients $f_t' = -(\partial/\partial y)\overline{F}_t(z, y)/\overline{F}_t$ and $s_t' = -(\partial/\partial y)\overline{S}_t(z, y)/\overline{S}_t$ measure the relative marginal fecundity and survival costs of phenotypic adjustment at age t per unit of phenotype change. Larger positive values of f_t' and s_t' reflect stronger fecundity and viability selection against phenotypic adjustment. Throughout, the use of an overbar, as in $\overline{F}_t(z, y)$ and $\overline{S}_t(z, y)$, will signify an average across environmental states (e.g., $\overline{F}_t(z, y) = \sum_{e=\mathrm{A,B}} \mathrm{P}[E_t = e] \times F_t^e(z, y)$). The selection coefficients f_t' and s_t' depend only on these averages as a result of our assumption that the marginal costs of phenotypic adjustment do not differ between environmental states A and B.

If selection is weak, the difference in reproductive success between the life history of an individual with reaction norm h and the reference life history can be approximated by a linear function in the selection coefficients. In order to minimize the approximation errors in this step of the analysis, we choose the reference phenotype z equal to the value that maximizes lifetime reproductive success for an individual with a fixed phenotype. Using once more the recursive definition of expected future reproductive success ($R_t = S_t(F_t + R_{t+1})$), the relative fitness advantage of a phenotypically plastic individual can now be expressed in terms of its additional reproductive success from age t onward, $\partial\overline{R}_t$, relative to an individual with the fixed phenotype z.

The fitness measure $\partial\overline{R}_t$ is a function of the state of the individual at age t after it has observed the state of the environment and updated its estimate of p_t but before it has adjusted its phenotype (indicated by the block arrow in fig. 1). Based on the derivation in the appendix, $\partial\overline{R}_t$ is defined by a sum of three terms that correspond to three subsequent steps in the cycle of events that occur in each breeding season:

$$\begin{aligned}\partial\overline{R}_t(x_{t-1}, p_t) = & -|h_t(x_{t-1}, p_t)|\left(s_t' + \frac{\overline{F}_t\overline{S}_t}{\overline{R}_t}f_t'\right) \\ & + (x_{t-1} + h_t(x_{t-1}, p_t) - z)\sum_{e\in\{\mathrm{A,B}\}}\mathrm{P}[E_t = e|\boldsymbol{O}_t = \boldsymbol{o}_t] \\ & \times\left(s_t^e + \frac{\overline{F}_t\overline{S}_t}{\overline{R}_t}f_t^e\right) + \left(1 - \frac{\overline{F}_t\overline{S}_t}{\overline{R}_t}\right)\sum_{o\in\{\mathrm{A,B}\}}\mathrm{P}[O_{t+1} = o|\boldsymbol{O}_t = \boldsymbol{o}_t] \\ & \times \partial\overline{R}_{t+1}(x_{t-1} + h_t(x_{t-1}, p_t), \mathrm{U}(p, o)).\end{aligned} \tag{5}$$

First, the organism changes its phenotype from the old value x_{t-1} to the new value $x_t = x_{t-1} + h_t(x_{t-1}, p_t)$, at which point it has to pay the cost of plasticity. The resulting fitness reduction, captured by the first term on the right-hand side above, is proportional to the amount of phenotypic adjustment and increases with the marginal fecundity and survival costs of plasticity at age t, f_t', and s_t'. These two costs are weighted according to their relative impact on the remaining lifetime reproductive success: reduced fecundity only affects the expected reproductive output in the current season (its relative contribution to the remaining reproductive success is given by $\overline{F}_t\overline{S}_t/\overline{R}_t$), whereas reduced survival impacts all further reproductive success from age t onward (a similar differential weighting applies to the coefficients f_t^e and s_t^e, discussed in the following paragraph).

After phenotypic adjustment, the organism is first subject to viability selection and then to fecundity selection. Accordingly, the second term on the right-hand side of equation (5) measures the fitness effect of expressing the new phenotype x_t (written as $x_{t-1} + h_t(x_{t-1}, p_t)$), relative to the fitness of the reference individual with phenotype z. The magnitude of this contribution to $\partial\overline{R}_t$ depends on the difference between x_t and z, as well as on the fitness gradient (given by the term $s_t^e + f_t^e\overline{F}_t\overline{S}_t/\overline{R}_t$) averaged over the distribution of environmental states across the individuals with observation history $\boldsymbol{O}_t = \boldsymbol{o}_t$ (here and elsewhere, $\boldsymbol{O}_t = \boldsymbol{o}_t$ stands for the composite event $O_t = o_t \cap O_{t-1} = o_{t-1} \cap \ldots \cap O_1 = o_1$). By definition (1), the distribution of environmental states for such individuals is given by $\mathrm{P}[E_t = \mathrm{A}|\boldsymbol{O}_t = \boldsymbol{o}_t] = p_t$ and $\mathrm{P}[E_t = \mathrm{B}|\boldsymbol{O}_t = \boldsymbol{o}_t] = 1 - p_t$, which captures the critical connection between an individual's estimate of the state of the environment and the selective conditions that it is likely to experience.

The final step in each cycle of events is the transition from the current breeding season to the next, which is

associated with a potential change in the state of the environment, a new observation $O_{t+1} = o$, and an update of the estimate p_t to $p_{t+1} = \mathrm{U}(p_t, o)$. The final term on the right-hand side of equation (5) takes into account that individuals can be in two different states after these events, depending on their observation at age $t + 1$. The contribution of each of the corresponding future life-history trajectories to the remaining lifetime reproductive success is weighted by its probability of occurring, and the entire sum is multiplied by the relative contribution of future fitness to the current remaining reproductive success, $1 - \overline{F}_t\overline{S}_t/\overline{R}_t$. According to equation (A18) in the appendix, the probabilities $\mathrm{P}[O_{t+1} = \mathrm{A}|\boldsymbol{O}_t = \boldsymbol{o}_t]$ and $\mathrm{P}[O_{t+1} = \mathrm{B}|\boldsymbol{O}_t = \boldsymbol{o}_t]$ can again be expressed in terms of p_t.

The final step in the linearization procedure is to approximate equation (4) for the long-term average fitness of the reaction norm, using the fact that all $\partial\overline{R}_t$ are small. Under this approximation, the optimization task reduces to maximizing the relative difference ∂W in expected lifetime reproductive success between a plastic individual and an individual with the optimal fixed phenotype z, where ∂W is given by

$$\partial W = \frac{W - \overline{R}_1}{\overline{R}_1} \approx \sum_{o \in \{\mathrm{A},\mathrm{B}\}} \mathrm{P}[O_1 = o]\partial\overline{R}_1(x_0, \mathrm{U}(p_0, o)). \quad (6)$$

As indicated by equation (5), the maximization of ∂W requires the optimization of a sequence of interdependent functions $\partial\overline{R}_t$. Since the dependency between these functions is unidirectional according to equation (5), the optimal reaction norm h can be found by backward state-dependent optimization. That is, we first maximize $\partial\overline{R}_T$, then $\partial\overline{R}_{T-1}$, and so on until $\partial\overline{R}_1$ has been maximized. The final step of the optimization is to find the optimal initial phenotype x_0. An annotated version of the C++ code used for the optimization is available from the Dryad Digital Repository: http://dx.doi.org/10.5061/dryad.00nd1 (Fischer et al. 2013).

Results

Optimal Reaction Norms for a Semelparous Life History

In order to calculate the optimal reaction norm h_t, it is necessary to specify the life history of the organism, as determined by the fecundity and survival probability functions $F_t^{e_t}(x_t, \Delta x_t)$ and $S_t^{e_t}(x_t, \Delta x_t)$. A simple case, which we will consider first, is when the species is semelparous, meaning that individuals reproduce once after reaching maturation at age T and die afterward. Lifetime reproductive success then depends on the cumulative survival up to the reproductive event and the organism's fecundity. We assume that only survival is affected by the phenotype in each respective environment and take

$$S_t^{e_t}(x_t, \Delta x_t) = \begin{cases} 1 - s(1 - x_t) - c\Delta x_t & \text{if } e_t = \mathrm{A}, \\ 1 - sx_t - c\Delta x_t & \text{if } e_t = \mathrm{B}. \end{cases} \quad (7)$$

Accordingly, at all ages, the optimal phenotype in environment A is $x_t = 1$, whereas $x_t = 0$ is optimal in environment B. The parameter s ($0 < s \ll 1$) determines the survival disadvantage of maladapted phenotypes and, therefore, measures the strength of selection. In addition, survival at each time step decreases with the current amount of phenotypic adjustment. Parameter c ($0 < c \ll 1$) measures the cost of plasticity, which we assume to be independent of the state of the environment. For the fecundity function, we take $F_t^{e_t}(x_t, \Delta x_t) = 0$ for all $0 < t < T$. The fecundity at age T, $F_T^{e_t}(x_T, \Delta x_T) = \varphi$, is independent of e_T, x_T, and Δx_T, with $\varphi > 1$ set by density dependence, such that the population remains stationary.

With these definitions, the recursion for the expected net fitness effect of plasticity, $\partial\overline{R}_t$ (eq. [5]), simplifies to

$$\begin{aligned} \partial\overline{R}_t(x_{t-1}, p_t) = {} & -c|h_t(x_{t-1}, p_t)| + s(2p_t - 1) \\ & \times (x_{t-1} + h_t(x_{t-1}, p_t) - z) \\ & + \sum_{o \in \{\mathrm{A},\mathrm{B}\}} \mathrm{P}[O_{t+1} = o|\boldsymbol{O}_t = \boldsymbol{o}_t]\partial\overline{R}_{t+1} \\ & \times (x_{t-1} + h_t(x_{t-1}, p_t), \mathrm{U}(p_t, o)). \end{aligned} \quad (8)$$

This expression is accurate up to first order in s and c (appendix). The first two lines on the right-hand side quantify the current cost and benefit of phenotypic adjustment, whereas the terms on the third and fourth lines take into account its future fitness consequences. If the organism has no or little information about the state of the environment ($p_t \approx 1/2$), then current survival is maximized if no phenotypic adjustment occurs (i.e., the cost term is minimized by $h_t = 0$). However, when the absolute value of $s(2p_t - 1)$ exceeds c, it becomes beneficial to adjust the phenotype to either $x_t = 1$ or $x_t = 0$, depending on what the current state of the environment is estimated to be.

As explained in the previous section, the estimate p_t changes in response to the sequence of observations made by the individual, according to the Bayesian update rule (2). Consider, for example, an individual with maturation age $T = 6$ who makes the observations $\boldsymbol{o} = (\mathrm{B}, \mathrm{B}, \mathrm{B}, \mathrm{A}, \mathrm{A}, \mathrm{A})$ during its life. In an environment with switching rates $\alpha = 0.15$ and $\beta = 0.1$ and sampling accuracy $a = 0.7$ (the parameters used earlier for illustrating eq. [2]), the estimate of the focal individual changes from $p_0 = 0.6$ to $p_1 = 0.39$, $p_2 = 0.25$, $p_3 = 0.18$, $p_4 = 0.48$, $p_5 = 0.71$, and $p_6 = 0.83$ (this sequence is indicated by the gray lines and circles in the left panel of fig. 2*a*).

In accordance with the preceding discussion of equation (8), we find that individuals with the optimal reaction norm (found for $s = 0.05$ and $c = 0.02$ by backward

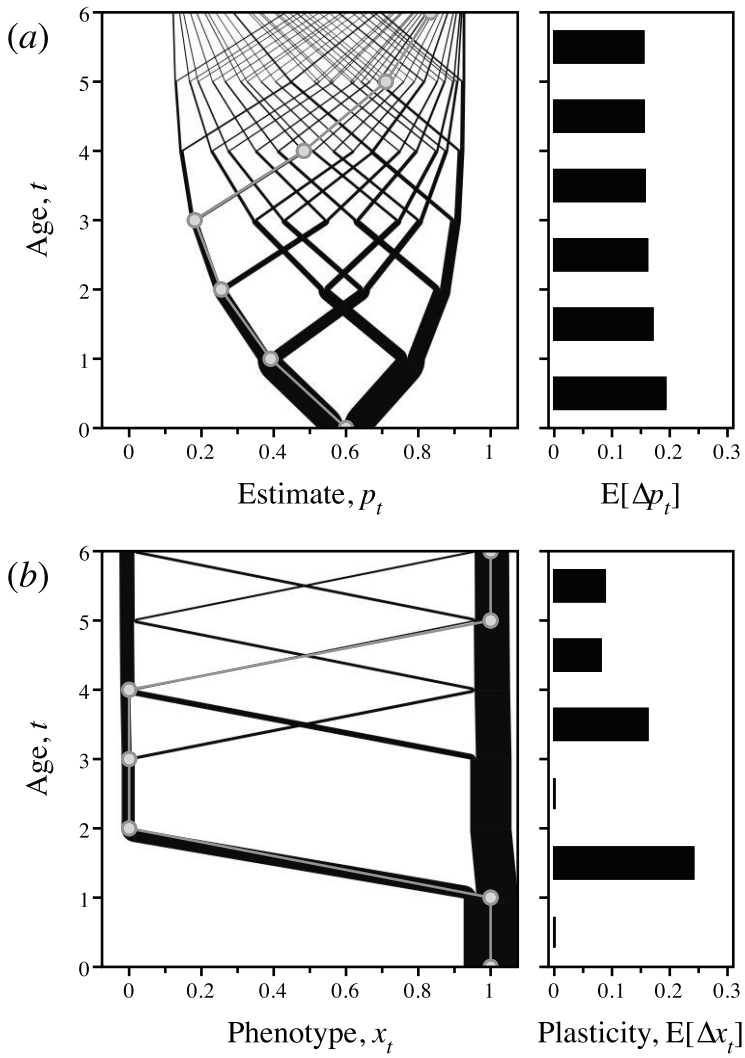

Figure 2: Estimated environmental conditions and resultant plastic phenotypes for a semelparous life history. *Left column*, Gray lines and circles indicate the estimated environmental conditions p_t (*a*) and the resultant phenotypes x_t (*b*) throughout the lifetime of a single individual that is making the sequence of observations $\boldsymbol{o} = (\mathrm{B}, \mathrm{B}, \mathrm{B}, \mathrm{A}, \mathrm{A}, \mathrm{A})$; black lines show the tree of estimates (*a*) and the tree of phenotypes (*b*) characterizing the ensemble of many individuals, each experiencing its own personal sequence of observations for a randomly drawn realization of the sequence of environmental states. The likelihood of a particular estimate or phenotype occurring along the tree is proportional to line thickness and depends on the rate of environmental fluctuations and on the sampling certainty. *Right column*, Bars indicate the absolute change of p_t (*a*) and x_t (*b*) averaged over the distribution of all observation sequences. For this example, the optimal reaction norm leads to plasticity at age 2 and during the second half of life. Parameters: $T = 6$, $\alpha = 0.15$, $\beta = 0.1$, $a = 0.7$, $s = 0.05$, and $c = 0.02$.

state-dependent optimization) switch between $x_t = 1$ and $x_t = 0$ only when they are sufficiently confident that their current phenotype is suboptimal under the present environmental conditions. For the example individual with observation sequence $\boldsymbol{o} = (\mathrm{B}, \mathrm{B}, \mathrm{B}, \mathrm{A}, \mathrm{A}, \mathrm{A})$, this means that the phenotype switches from $x_1 = 1$ to $x_2 = 0$ after the individual observes for the second time that the environment is in state B (when its estimate is $p_2 = 0.25$). At a later stage, the phenotype switches back again from $x_4 = 0$ to $x_5 = 1$ after state A has been observed twice, first at age 4 and then at age 5 (the estimate is then $p_5 = 0.71$). In both cases, the switching points are correctly predicted by the condition $s|2p_t - 1| > c$ (but see discussion on the time dependency of the reaction norm below). The phenotype trajectory $x_0 \to x_1 \to \ldots \to x_6$ for the example individual is highlighted in figure 2*b* (left part; gray lines and circles).

So far, we have focused on a single observation se-

quence. With $T = 6$, there are $2^6 = 64$ possible sequences of observations, which collectively give rise to a bifurcating tree of estimate and phenotype trajectories (shown in black in the left column of fig. 2). Which path through the tree an individual will take is determined by its sequence of observations: each branch in the tree of estimates (fig. 2*a*) splits into two new branches at the next observation event, from which the individual will follow the right path if it observed A or the left path if it observed B. Accordingly, the rightmost and leftmost paths in the tree correspond to the observation sequences (A, A, A, A, A, A) and (B, B, B, B, B, B), respectively. The phenotype tree (fig. 2*b*) does not necessarily split after each observation because the optimal reaction norm induces a phenotypic switch only when the individual is sufficiently confident that its current phenotype is suboptimal.

In general, not all observation sequences have the same probability of occurrence. First, if the environment is strongly autocorrelated and the sampling accuracy is high, sequences with no or very few switches, such as (A, A, A, A, A, A), will be much more likely to occur than sequences with many switches, such as (A, B, A, B, A, B). This effect is visible to some extent in figure 2*a*, where the likelihood that a particular path occurs is indicated by its line width relative to that at the root of the tree. Paths in the interior of the tree in figure 2*a* are less likely than paths with fewer switches that lie on the outside. This pattern becomes more pronounced at higher sampling accuracy and lower rates of switching (not shown). A second asymmetry is caused by unequal switching rates, which bias the weights of paths along the estimate tree toward the environmental state that is more frequent. In figure 2*a*, this effect reveals itself by the slightly increased thickness of paths in the right part of the tree.

The phenotype tree (fig. 2*b*) is generally highly asymmetric because the optimal initial phenotype for a naive individual, x_0, is adapted to the most likely environmental state (in this case, state A). This is the typical outcome if the survival and fecundity functions are linear and the two environmental states are not equally frequent. As indicated by the relative thickness of the terminal branches of the phenotype tree, the initial phenotype has a prolonged effect on the phenotype distribution: at the final age T, $\beta/(\alpha + \beta) = 40\%$ of the individuals are in an environment in state B, but the optimal reaction norm induces less than 30% of the individuals to actually exhibit the phenotype $x_T = 0$ adapted to this state. The reason is that some individuals in environment B made observation errors, preventing them from adjusting their phenotype from its initial value $x_0 = 1$.

To quantify the rate of information accumulation and the degree of plasticity at various ages, we calculated the absolute change in forecasting probabilities $\Delta p_t = |p_t - p_{t-1}|$ and phenotypes $\Delta x_t = |x_t - x_{t-1}|$ for all sequences of observations and averaged these values across the tree, weighting by the likelihood of each observation sequence across all possible realizations of the environment. The rate of information accumulation decreases monotonically with age (fig. 2*a*, *right panel*) before it asymptotes toward a stable level. This shows that organisms become better at estimating environmental states the more often they sample, although they are limited in the level of certainty they can achieve. Phenotypic plasticity (measured as $E[\Delta x_t]$; fig. 2*b*, *right panel*) reaches a maximum in the second season and decreases over the final three seasons. For the parameters considered in figure 2, no individuals adjust their phenotype in the first or the third season.

To illustrate the structure of the optimal reaction norm, we maximized equation (8) while treating p_t as a continuous state variable (in reality, p_t can only take a discrete set of values, one for each possible observation sequence). The resulting representation of the optimal reaction norm h_t (fig. 3 shows results for h_6) reveals three regions in state space with qualitatively different optimal responses. First, there is a plateau at intermediate levels of p_t, where the optimal adjustment $h_t(x_{t-1}, p_t)$ is zero. This indicates that organisms have to acquire a particular level of certainty about environmental conditions before they adapt their phenotype. When the estimate p_t lies either to the left or to the right of the plateau, it is beneficial to adjust the phenotype. If the fitness function is linear, then it is always optimal to change to either $x_t = 0$ (at low values of p_t) or $x_t = 1$ (at high values). In figure 3, indicated by black dots and curves, respectively, are the states and the transitions between states of the example individual from figure 2. Note that multiple, consistent observations are necessary to traverse the plateau and enter the region of phenotypic adjustment, helping to buffer the organism against observation errors.

The width of the plateau at age T is equal to c/s (see appendix), and phenotypic adjustment occurs only if $p_t < (1 - c/s)/2$ or $p_t > (1 + c/s)/2$. Therefore, as one would expect, phenotypic adjustment becomes less likely if the cost of plasticity, c, is high or if the benefit of expressing an adapted phenotype, s, is low. The plateau disappears if $c = 0$. On the other hand, if $c > s$, then organisms never adjust their phenotype in their final season, although they may still do so earlier in life. In line with this result, we observe the optimal reaction norm to depend on time. The width of the plateau is maximal at $t = T$ (for comparison, dashed lines in fig. 3 outline the contours of h_1), such that there are states close to the edges of the plateau for which organisms adjust their phenotype when they are young but not when they are older.

The time dependency of the reaction norm is strongest at the end of life, when it is necessary to compensate for

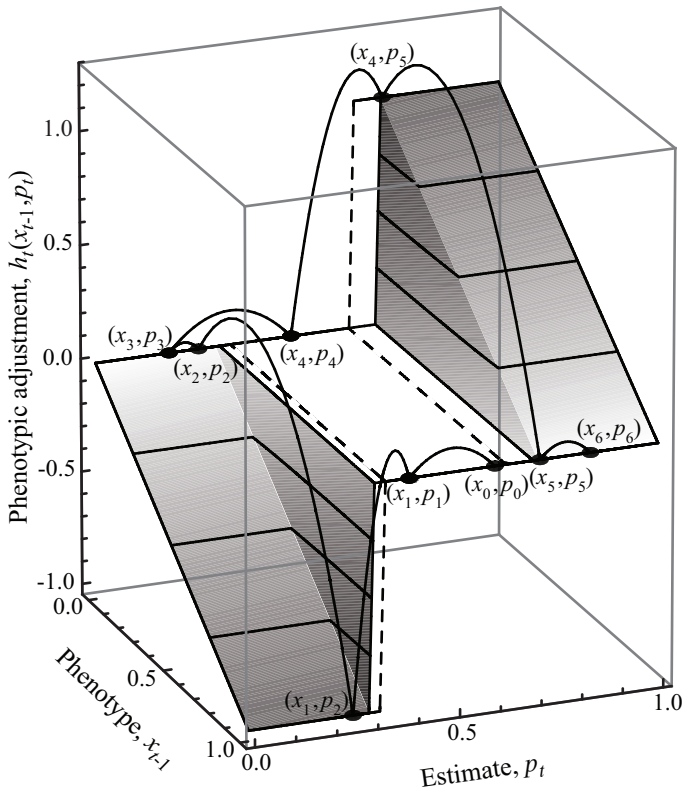

Figure 3: Optimal reaction norm, describing the phenotypic adjustment $h_t(x_{t-1}, p_t)$ as a function of the current estimate p_t of environmental conditions and the previous phenotype x_{t-1}. The shaded surface depicts the optimal reaction norm h_6 at age 6, while the dashed lines outline the optimal reaction norm h_1 at age 1. The plateau at intermediate values of p_t applies to individuals that are not sufficiently certain about the state of the environment to adjust their phenotypes. This plateau is flanked by two ranges of conditions under which individuals change their phenotype to either $x_t = 0$ (left-hand side, where p_t is low) or $x_t = 1$ (right-hand side, where p_t is high). The width of the plateau equals c/s at age T (appendix, available online), being more narrow at younger ages. Filled circles connected by curved lines indicate the change of state variables for the particular individual shown in figure 2, which is making the sequence of observations $\boldsymbol{o} =$ (B, B, B, A, A, A). Parameters are as in figure 2.

the reduced levels of plasticity in the final life stages (particularly if $c > s$). However, these compensatory effects dampen out generally within a few backward optimization steps, such that the reaction norms at early ages are indistinguishable in practice. The biological implication is that end-of-life-effects on patterns of plasticity are likely to be confined to the last few stages of an individual's life history.

Depending on how organisms update their estimate p_t after each observation and how wide the plateau of the reaction norm is, the optimal reaction norm can be associated with a variety of realized phenotype sequences and resulting patterns of plasticity. Figure 4 illustrates the main effects of the various parameters of the model. In stable environments (fig. 4*a*, left), individuals adjust their phenotype early in life once they have become sufficiently confident that their initial phenotype is suboptimal. Trait reversal later in life is rare. The frequency of reversal to the initial phenotype goes up as the rate of environmental fluctuations increases, leading to a high average amount of phenotypic adjustment at intermediate values of α and β (data not shown). Yet, in highly variable environments (fig. 4*a*, middle), the organism cannot always build up a confident estimate before the environment switches again, and any phenotypic adjustments that do occur are likely to be beneficial for only a short time. Hence, the overall level of plasticity decreases once the inherent unpredictability of the environment starts to limit the future benefits of phenotypic adjustment. In the example shown in the middle panel of figure 4*a*, we still find a plasticity window in the midlife period, when the expected future benefits of phenotypic adjustment are still considerable and when at least a small subset of the organisms have made a series of consistent observations justifying an adjustment of the phenotype.

The amount of sampling that is needed to establish the

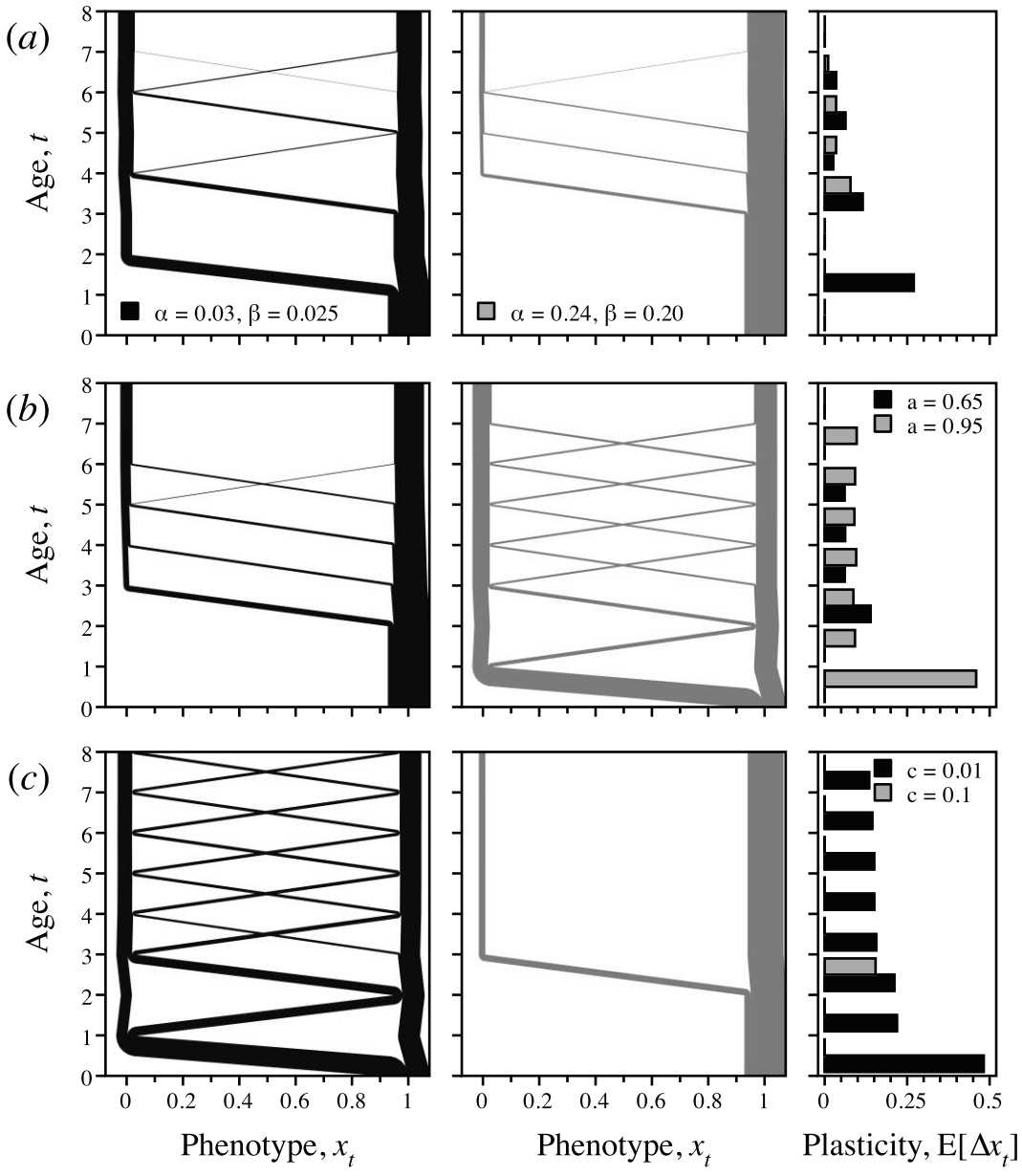

Figure 4: Dependence of optimal plasticity patterns on model parameters. Comparisons between optimal patterns of plasticity in environments with rare transitions (approximately once every four lifetimes; lines in left column) and frequent transitions (approximately twice per lifetime; gray lines in middle column) between the two environmental states (*a*), between low (black lines in left column) and high (gray lines in middle column) sampling certainty (*b*), and between low (black lines in left column) and high (gray lines in middle column) cost of plasticity (*c*). As in figure 2*b*, the right column shows the absolute phenotypic adjustment, averaged over the distribution of all observation sequences, with black bars corresponding to the left column and gray bars to the middle column. Parameters, where not indicated otherwise: $T = 8$, $\alpha = 0.12$, $\beta = 0.1$, $a = 0.7$, and $s = c = 0.05$.

current state of environment with a sufficient level of confidence is determined by the sampling accuracy. If observation errors are rare (fig. 4*b*, middle), a single observation can be enough to trigger a phenotype change, whereas at lower sampling accuracy, organisms maintain their initial phenotype for a while before they start to specialize (fig. 4*b*, left). Moreover, once specialized, individuals rarely reverse their phenotype. These results are explained by the fact that the sampling accuracy is related to how much the estimate p_t changes after an observation (eq. [2]). The

estimate changes in small steps if the sampling accuracy is low, such that it may take several consistent observations to traverse the plateau of the reaction norm and enter the region of state space where phenotypic adjustment is beneficial. By contrast, when the sampling accuracy is high, the change in p_t induced by an observation can be sufficient to jump over the plateau in one step, leading to an immediate adjustment of the phenotype after each observation.

Similar effects are observed by varying the cost of phenotypic adjustment (fig. 4*c*). If adjusting the phenotype is costly (fig. 4*c*, middle), the plateau of the reaction norm is wider, such that traversing the plateau requires a larger number of consistent observations (equivalent to decreasing the sampling accuracy). Conversely, if the cost of plasticity is low (fig. 4*c*, middle), the plateau is easily traversed in a single step, analogous to the situation at high sampling accuracy.

Iteroparous Life Histories with Fecundity or Viability Selection

Our main result for the fitness consequences of phenotypic adjustment (eq. [5]) suggests that the life history of an organism strongly influences its optimal plasticity schedule. For example, a combination of life-history parameters appears as a factor $1 - \overline{F}_t\overline{S}_t/\overline{R}_t$ in front of the expected future fitness effect on the third line of equation (5). Life-history differences, therefore, affect the relative weighting of current and future consequences of plasticity. Furthermore, this weighting is different depending on whether the costs and benefits of plasticity act on fecundity or on survival (the fecundity effects f_t' and f_t^e are preceded by a factor $\overline{F}_t\overline{S}_t/\overline{R}_t$, which reflects the relative importance of current reproduction).

To quantify the effects of life history on plasticity, we introduce a heuristic measure I_t that captures how important the immediate effects of phenotypic adjustment are relative to their effects on future fitness components in the calculation of lifetime reproductive success (eq. [5]). Our definition is as follows:

$$I_t = \frac{s_t' + \bar{s}_t + (\overline{F}_t\overline{S}_t/\overline{R}_t)(f_t' + \bar{f}_t)}{s_t' + \bar{s}_t + (\overline{F}_t\overline{S}_t/\overline{R}_t)(f_t' + \bar{f}_t) + (1 - [\overline{F}_t\overline{S}_t/\overline{R}_t])(s_t' + f_t' + \bar{s}_t + \bar{f}_t)}, \quad (9)$$

where $\bar{f}_t = (\alpha|f_t^{\mathrm{A}}| + \beta|f_t^{\mathrm{B}}|)/(\alpha + \beta)$ and $\bar{s}_t = (\alpha|s_t^{\mathrm{A}}| + \beta|s_t^{\mathrm{B}}|)/(\alpha + \beta)$ represent the average strength of fecundity and viability selection at age t across environments. The value of I_t lies between 0 and 1, with $I_t = 0$ corresponding to a situation in which current phenotypic adjustments have no consequences for lifetime reproductive success (this may occur when the cost and benefit of plasticity manifest themselves in the form of fecundity selection and current fecundity is negligible relative to the expected reproductive fitness in the future) and with $I_t = 1$ indicating that only current reproductive success is relevant to the optimization of the reaction norm (as, e.g., at $t = T$). Accordingly, we refer to I_t as the impact of current phenotypic adjustment on the remaining lifetime reproductive success.

Low values of I_t are expected to favor delayed phenotypic adjustment, for the reason that postponing plasticity has limited consequences for current reproductive success, whereas it will allow for additional observations before the organism commits to a costly phenotypic change. Given that I_t increases monotonically with $\overline{F}_t\overline{S}_t/\overline{R}_t$, we expect that in iteroparous life histories, plasticity will be concentrated at those ages where individuals realize a large fraction of their lifetime reproductive success. Furthermore, this bias is predicted to be more pronounced if the cost and benefit of plasticity are mediated by effects on fecundity (as opposed to survival, as we have thus far assumed).

To illustrate these predictions, we calculated the optimal reaction norm for an example iteroparous life history based on published data from a life-table response experiment using the estuarine polychaete *Streblospio benedicti* (Levin et al. 1996; fig. 5). *Streblospio benedicti* occupies soft mucoid sediment tubes from where it feeds either by extending its tentacles up into the water column or by sweeping its feeding palps across the sediment surface. We will therefore consider feeding mode as a potentially plastic phenotype that we will assume to be under divergent selection across environmental states. In our calculations, the observed fecundity and survival parameters from the original life-table response experiment (φ_t and σ_t; specified in table A1, available online, and plotted in fig. 5) were modified by (hypothetical) costs of feeding-mode adjustments and the fitness advantage of expressing an adapted foraging strategy. We considered two scenarios for this iteroparous life history, labeled as "viability selection" (fig. 6*a*) and "fecundity selection" (fig. 6*b*). In addition, we calculated the optimal reaction norm for a comparable semelparous life history (fig. 6*c*), using identical values for the parameters T, α, β, a, s, and c.

For the viability selection scenario, we assumed that all fitness effects of plasticity manifested themselves as changes in survival. The fecundity and survival functions were defined by

$$F_t^{\mathrm{A}}(x_t, \Delta x_t) = F_t^{\mathrm{B}}(x_t, \Delta x_t) = \varphi_t \quad (10a)$$

and

$$S_t^{e_t}(x_t, \Delta x_t) = \begin{cases} \sigma_t \exp[-s(1 - x_t) - c\Delta x_t] & \text{if } e_t = \mathrm{A}, \\ \sigma_t \exp(-sx_t - c\Delta x_t) & \text{if } e_t = \mathrm{B}. \end{cases} \quad (10b)$$

The optimal phenotype tree under these conditions (fig. 6*a*, *left panel*) is difficult to distinguish from the result for

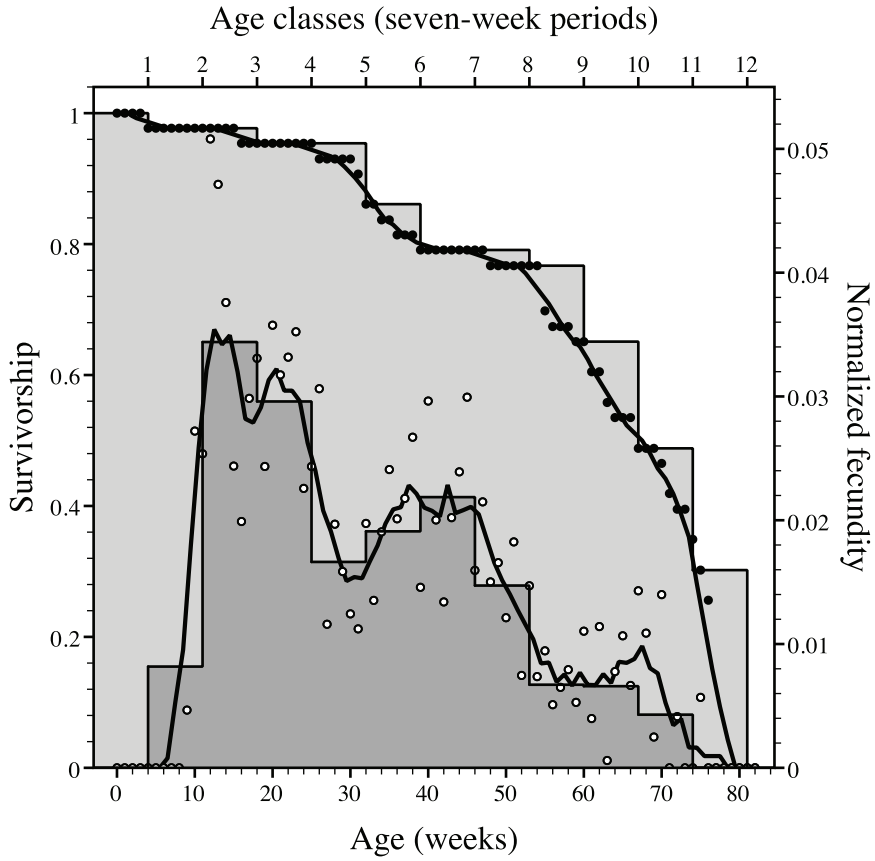

Figure 5: Age-specific fecundity and survivorship schedules for *Streblospio benedicti*. Data points show the survivorship (filled circles) and weekly fecundity (open circles; normalized so as to yield a lifetime reproductive success of 1) observed in the control treatment of a life-table response experiment with the estuarine polychaete *S. benedicti* (Levin et al. 1996; data were collected for a cohort of 50 individuals). Lines show the observed survivorship smoothed over a 3-week period using least squares smoothing (figs. 2, 3; Levin et al. 1996). Before using these empirical observations to parameterize our model, we first partitioned the data into 12 age classes and calculated the expected survival and fecundity over each of the resulting 7-week periods (values provided in table A1, available online). The thus-determined survivorship and normalized fecundity are presented as dark gray and light gray histograms, respectively, and have retained the main features of the original data.

the semelparous history (fig. 6*c*, *left panel*): small differences in the expected amount of phenotype change occur from age 5 onward (fig. 6*a*, 6*c*, *right panels*). These findings are consistent with the impact profiles I_t of the two life histories (fig. 6*a*, 6*c*, *center panels*), which are overall comparable, except for the final age classes, where I_t for the iteroparous life history increases as a result of the decline of fecundity rates toward the end of life.

The fecundity and survival schedules in the fecundity selection scenario were defined as:

$$F_t^{e_t}(x_t, \Delta x_t) = \begin{cases} \varphi_t \exp\left[-s(1 - x_t)\right] & \text{if } e_t = \text{A}, \\ \varphi_t \exp\left(-sx_t\right) & \text{if } e_t = \text{B}, \end{cases} \quad (11a)$$

and

$$S_t^{\text{A}}(x_t, \Delta x_t) = S_t^{\text{B}}(x_t, \Delta x_t) = \sigma_t \exp\left(-c\Delta x_t\right), \quad (11b)$$

such that the costs of plasticity reduced survival, while the expression of an adapted phenotype was favored by fecundity selection. In this case, as reflected by the impact profile, plasticity provides limited benefits before the organism has actually started to reproduce, leading to a delay in the onset of plasticity relative to the semelparous life history (fig. 6*b*, 6*c*). Also in this case, a comparison of the impact profiles explains the main differences between the plasticity schedules of the iteroparous and the semelparous life histories. However, without a base for comparison, the impact profile is a poor predictor of the absolute levels of phenotypic adjustment, because the schedule of plasticity is affected primarily by the dynamics of information accumulation. For instance, even in figure 6*b*, there is a peak of plasticity early in life at the onset of reproduction, when the impact I_t is still relatively low.

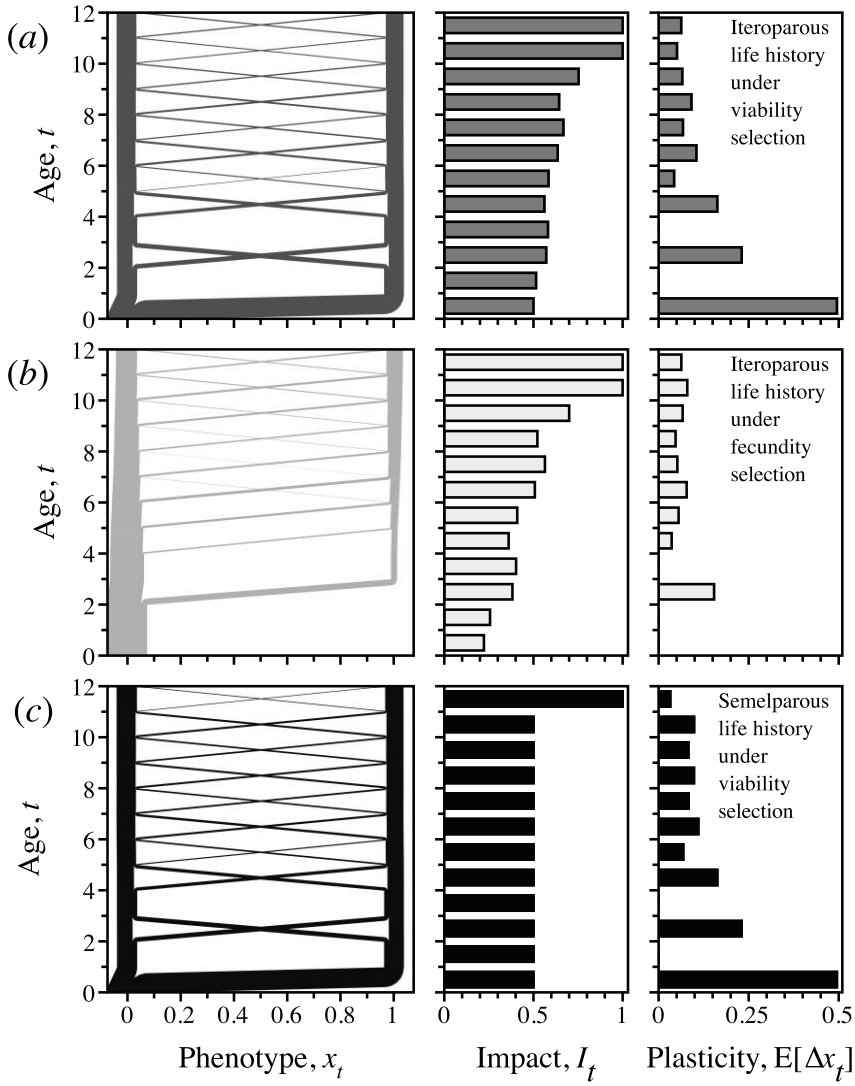

Figure 6: Dependence of optimal plasticity patterns on life-history types and selection regimes. The left column shows trees of phenotypes resulting from the optimal reaction norm for an iteroparous life history (*a*, *b*) and a semelparous life history (*c*). Adapted phenotypes benefit from either reduced mortality (*a*, *c*; viability selection) or increased fecundity (*b*; fecundity selection). The central and right columns show, respectively, the relative importance of immediate and future fitness effects, measured by the impact I_t of current phenotypic adjustment and the average absolute phenotypic adjustment for the phenotype trees in the left column. Parameters: $T = 12$, $\alpha = 0.081$, $\beta = 0.086$ (i.e., the environment switches between states on average once per 12 time steps), $a = 0.667$, $s = 0.05$, and $c = 0.02$. Using the definition in the main text, the impact of current phenotypic adjustment is calculated as follows for $t < T$: $I_t = 1/2$ (*a*), $I_t = R_t/(2R_t - \varphi_t\sigma_t)$ (*b*), and $I_t = (cR_t + s\varphi_t\sigma_t)/[(2c + s)R_t - c\varphi_t\sigma_t]$ (*c*). In all three cases, $I_T = 1$. The life-history parameters φ_t, σ_t, and R_t for the iteroparous life history are listed in table A1, available online.

Discussion

The responsiveness of phenotypically plastic organisms to cues from the environment often varies with age. Various empirically observed patterns of age-dependent plasticity have been suggested to result from changes in the availability, reliability, and usefulness of environmental information over the course of an individual's life (Dufty et al. 2002). To formally evaluate this idea, we have modeled the developmental trajectory of an organism living in a stochastically fluctuating environment, about which the organism obtains information by sampling at regular intervals throughout its life. The evolutionarily optimal response for such an organism is to adjust its phenotype only if it is sufficiently confident of the current state of the environment. Accordingly, for linear and certain nonlinear (fig. A1*b*) fitness functions, a characteristic feature

of the optimal reaction norm is that it has a plateau at intermediate values of the state variable p_t, which represents the organism's current estimate of the state of the environment (fig. 2). The width of the reaction norm's plateau is dependent on the ratio between the cost of phenotype adjustment and the benefit of expressing the optimal phenotype, c/s. In our model, the dynamic of an individual's state in the state space spanned by the optimal reaction norm h_t is specified by a Bayesian update rule (2), which takes into account the reliability of a single observation, measured by the sampling accuracy a, and the inherent uncertainty of the environment, captured by the switching rates α and β. These parameters determine how many observations are needed for the estimate p_t to traverse the width of the plateau and, correspondingly, how quickly the organism will respond to a change in its environment.

According to our analysis, the interplay of environmental uncertainty and the accuracy of perceived information with life-history determinants and the fitness consequences of phenotypic adjustments must be expected to result in three distinct features of the pattern of age-dependent plasticity. First, the plateau of the reaction norm and the limited accuracy of perceived information typically cause a delay in the response of the organism to its environment, during which it integrates multiple observations into a sufficiently reliable estimate of the state of the environment. Moreover, older individuals take more time to respond to an environmental change during their lifetime than newborn individuals take to adjust their phenotype to the environmental condition at the start of their life. This is because newborn individuals have limited prior information about the state of the environment, whereas older individuals are biased by the information they have accumulated earlier. Correspondingly, a naive newborn individual starts sampling with a state located on the reaction norm's plateau and can therefore more easily be induced to adjust its phenotype than an older individual, whose estimate of the state of the environment, p_t, must generally first traverse at least the entire width of the plateau before a phenotypic adjustment will occur. Finally, we observed a reduction of plasticity toward the end of life under most parameter conditions and life histories we explored. This effect is particularly pronounced if the benefit of expressing the optimal phenotype is small relative to the cost of phenotypic adjustment, such that it does not pay to adjust the phenotype unless the individual can profit from this adjustment for several additional time steps before it dies. The combination of these early, mid-, and late-life effects can produce a variety of optimal age-dependent plasticity patterns, which are found to be nonmonotonic in general. A common pattern, which may be more or less pronounced depending on the parameters considered (cf. figs. 2*b*, 4, 6), features a (delayed) peak of phenotypic adjustments early in life (corresponding to the initial phenotypic adjustment by young individuals after they have accumulated sufficient information). After a period of reduced plasticity, the first peak of plasticity is followed by a second, broader one, which is caused by individuals responding to a change of the environment during their lifetime with the expectation that they will live long enough to benefit from a phenotypic adjustment.

To further explore the effects of life history on optimal patterns of age-dependent plasticity, we extended our analysis to an iteroparous example life history based on demographic data of the estuarine polychaete *Streblospio benedicti* (Levin et al. 1996). Phenotypes were exposed either to simulated viability or fecundity selection. Under viability selection, results for the iteroparous life history are similar to the predictions for a basic semelparous life history: the calculated optimal schedule of phenotypic switches is nearly identical (fig. 6*a*, 6*c*). If the fitness effects of phenotypic changes are mediated by differences in survival, it is risky to postpone phenotypic switches if they are beneficial, because both current and future reproductive success are conditional on current survival. By contrast, under fecundity selection, it is optimal to delay phenotypic adjustments until shortly before reproduction takes place, thus allowing for the accumulation of additional information and the subsequent maximization of the benefits of plasticity at the time of reproduction (fig. 6*b*; cf. fig. 5). Therefore, under fecundity selection, we would predict a single adjustment to the current environment at the penultimate time step before reproduction in a semelparous life history and a corresponding delay in plasticity until the onset of reproduction in an iteroparous life history (fig. 6*b*). These predictions need to be adjusted in situations where organisms use more than one season to accumulate the resources necessary for reproduction. As indicated earlier, a formal analysis of such cases needs to take into consideration additional state variables (e.g., the amount of energy reserves stored for reproduction). Although we have not performed this analysis, we expect that storing resources for reproduction would have similar effects for the expression of plasticity as shifting part of the reproductive output to earlier reproductive seasons; that is, plasticity would be expressed earlier in life.

An alternative mechanism also mentioned by Dufty et al. (2002) that could possibly be responsible for age-dependent plasticity are developmental constraints arising in the course of ontogeny. Developmental constraints would lead to increasingly canalized phenotypes while organisms pass through certain ontogenetic stages. Our model assumes that the range of attainable phenotypic states does not decrease with age. In this way, we could show that information gain can give rise to age-dependent changes of plasticity as an

emergent pattern without a priori introduction of hard constraints on the attainable range of phenotypes at different ages. Such constraints could, however, easily be included in our model to produce more detailed predictions. Dufty et al. (2002) proposed that later in life, information is only used for phenotypic fine-tuning, since developmental trajectories have already been fixed early in life. However, lifelong plasticity in leg length of barnacles (Marchinko 2003) is a counterexample to this suggestion. Further research is needed to clarify whether there are generalities in the way developmental constraints change during ontogeny. Our results show that, even without developmental constraints, plasticity later in life is generally expected to be lower than early in life.

Age dependence of phenotypic adjustment costs constitutes a third alternative mechanism that might cause age-dependent plasticity. Certain phenotypic responses induced during late ontogeny might cause greater (or smaller) costs than if induced early in life (Hoverman and Relyea 2007; Callahan et al. 2008). Likewise, sampling accuracy might change with age, for instance, because accuracy is enhanced over time by learning. Similar to developmental constraints, specific assumptions on age-dependent costs and sampling accuracy could readily be included in the model, but for the sake of simplicity and generality, we did not include them in this study. We also did not account for maintenance costs of plasticity in our model, which are associated with developing and maintaining the sensory and neural machinery necessary for processing environmental information (DeWitt 1998; Scheiner and Berrigan 1998; Van Buskirk and Steiner 2009; Auld et al. 2010). The magnitude and importance of maintenance costs of plasticity is debated. Recent empirical studies suggest that maintenance costs may be modest in the majority of cases (Van Buskirk and Steiner 2009). We expect that maintenance cost would influence the optimal level of plasticity but not otherwise affect the optimal pattern of age-dependent plasticity.

To our knowledge, no theoretical study has so far investigated possible mechanisms for the evolution of age-dependent plasticity. However, some theory exists on the evolution of reversible plasticity (Gabriel 1999; Gabriel et al. 2005). These studies investigated how lag times in phenotypic responses and the quality of an organism's environmental information affect optimal plasticity. Gabriel et al. (2005) considered the two extreme cases of complete information and no information gain through sampling only. Their analysis shows that nonspecialist phenotypes are superior to phenotypes that track environmental change if there is a lag in the phenotypic response, since lag times cause temporary maladaptation, reducing the fitness of plastic phenotypes. These models also predict that organisms should express less specialized phenotypes when information is incomplete than with perfect information, a result that is also supported by our findings. Our model adds a life-history perspective to the existing theory on the evolution of plastic responses by showing that plasticity can vary not only between different environments (Marchinko 2003; Relyea 2003) but also with age.

We are not aware of empirical studies that have tracked plastic adjustments throughout different individual life histories, so the predictions of our model can only be tested indirectly against empirically observed patterns of plasticity. For example, in barnacles *Balanus glandula*, wave action is highly correlated with leg length (Arsenault et al. 2001). Barnacle leg length is a plastic trait that responds very rapidly to new flow conditions. Hence, our model predicts that either the sampling accuracy must be high in this system or the cost-to-benefit ratio of plasticity c/s must be low. Barnacles are iteroparous, hermaphroditic, sessile organisms that reproduce several times per year. Nonadjusted leg length leads to a suboptimal food intake, which is likely to affect immediate reproductive success through viability or fecundity selection. Transitions between environmental states (high flow vs. low flow) are frequent (Arsenault 2001). In our model, the combination of these factors would tend to favor lifelong plasticity and frequent phenotypic adjustments, corresponding to the pattern observed in barnacles (Marchinko 2003). However, the degree of plasticity predicted by our model depends strongly on the adjustment cost, which has not yet been estimated empirically.

Another example is provided by the snail *Helisoma trivolvis*, which can adjust the size of its shell to the presence of predatory water bugs (*Belostoma flumineum*) in its environment (Hoverman and Relyea 2007). *Helisoma trivolvis* is an iteroparous species living in semipermanent ponds that can be colonized by water bugs at any time during development or adulthood. Adult water bugs and their nymphs are aquatic and prey on snails, so plasticity is likely to confer a viability selection advantage. Snails respond to the presence of predators by producing larger shells. Reversal of this trait is possible only in early development, whereas induction is possible for longer. Partial irreversibility of plasticity is predicted by our model when the environment is relatively stable and the sampling accuracy is low (e.g., fig. 4*b*). Yet, for *Helisoma*, developmental constraints are likely to play an important role as well because the shape of the shell cannot be altered once deposited (Hoverman and Relyea 2007).

Organisms living in a stochastically fluctuating environment with a limited ability to read environmental cues need to integrate current and past information in order to optimally adjust their phenotype to the state of the environment. We conclude that the accumulation of information during life and the optimal response of the

organism in the context of its life history are sufficient to produce striking patterns of age-dependent plasticity. Depending on the rate of environmental fluctuations, the accuracy of sampling, phenotypic adjustment costs, and the fitness component that is most strongly affected by selection (i.e., survival or reproduction), a diversity of age-dependent plasticity patterns can emerge. While these patterns correspond to the wide variety of plasticity schedules observed in nature and expressed across species, it is unlikely that these organisms use the exact complex Bayesian update rule assumed in our analysis. Instead, biological organisms often build on simple rules of thumb when navigating complex environments (Welton et al. 2003; McNamara and Houston 2009). Therefore, future research should explore whether there are simple decision rules for age-dependent plasticity that generate responses to stochastic environments that are similarly efficient to those generated by the rules assumed in our analysis.

Acknowledgments

We are grateful to J. Johansson, P. Taylor, and four anonymous reviewers for their valuable comments on various versions of the manuscript. This study was funded by the Austrian Science Fund (FWF; grant P18647-B16 to B.T.), the Swiss National Science Foundation (SNF; grant 31003A-133066 to B.T.), the Research Council of Norway (grant 214285 to B.F.), the Netherlands Organization for Scientific Research (NWO; VIDI grant 864.11.012 to G.S.v.D.), and the European Research Council (ERC; starting grant 30955 to G.S.v.D.). U.D. gratefully acknowledges financial support from the European Commission, the European Science Foundation, the FWF, the Austrian Ministry of Science and Research, and the Vienna Science and Technology Fund.

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